Letter to the Editor

Respiratory failure with hydrothorax due to acute onset pleuroperitoneal communication after upper endoscopy

Dear Editor,

Pleural effusion that results from pleuroperitoneal communication occurs in 1.6–10% of patients on continuous ambulatory peritoneal dialysis,[1,2] but is also infrequently observed in relation to liver cirrhosis.[3] We report an educational case of respiratory distress with hydrothorax due to acute onset of pleuroperitoneal communication after upper endoscopy.

A 67-year-old man with end-stage renal disease (on hemodialysis three times a week for over 7 years), hypertension, and liver cirrhosis (Child–Pugh B), was transferred to our hospital due to respiratory distress. He was in his usual state of health until 1 day prior to admission for routine upper endoscopic examination, which revealed no major disease and was completed without any acute complication.

On admission, his vital signs were stable except for slight tachypnea (respiratory rate 24 breaths/min) with desaturation (oxygen saturation 95% on 2 L/min oxygen administered by nasal cannula). Breath sounds had disappeared on the right side, but he was not edematous. Laboratory examination revealed significant changes reflecting underlying disease: platelet count, $6.8 \times 10^4/\mu L$; albumin, 2.3 mg/dL; and creatinine, 5.49 mg/dL. Chest radiographs showed a large right unilateral pleural effusion, which had changed drastically compared with 1 week prior (Fig. 1A,B). Computed tomography scan showed previously known mild ascites (Fig. 1C). Transthoracic echocardiogram showed normal cardiac function. The patient's history was indicative of iatrogenic esophageal perforation, however, this was

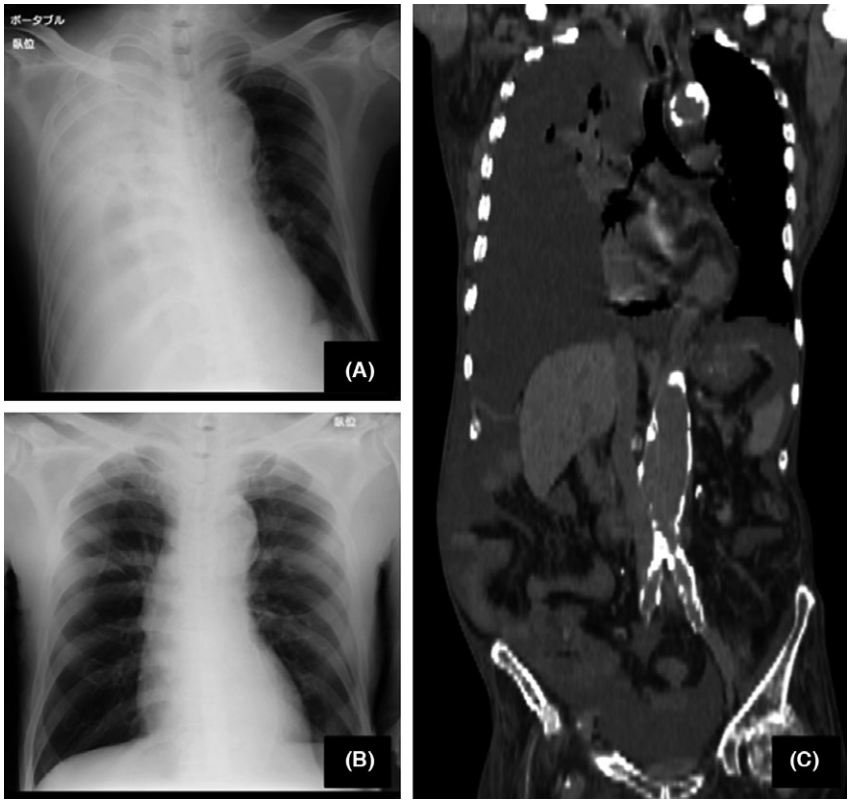

Fig. 1. A, Chest radiograph of a 67-year-old man with respiratory distress, taken at the time of admission, showing massive unilateral right pleural effusion. B, In contrast, a chest radiograph taken 1 week prior was almost normal, indicating that the massive pleural effusion had accumulated rapidly (at least within 1 week). C, Thoracoabdominal computed tomography showed previously known mild ascites in addition to right pleural effusion.

rigorously excluded by esophagography and upper endoscopy. Moreover, pleural fluid analysis revealed clear transudative fluid with negative culture studies and malignant cells. Taking these findings into consideration, hepatic hydrothorax was suspected. Although the direct proof[2,4] was not detected, the final diagnosis was hepatic hydrothorax due to pleuroperitoneal communication, because the biochemical nature of the pleural effusion and ascites was identical. The patient was moved to another hospital for subsequent management after thoracic drainage with a chest tube.

This case reveals two important clinical cautions for acute care physicians: (i) hydrothorax due to acquired pleuroperitoneal communication can sometimes occur suddenly in patients with ascites, (ii) a sudden increase in intra-abdominal pressure related to upper endoscopy (even with an intact vomiting reflex) may become a predisposing factor for the development of this condition. This situation is uncommon for acute care physicians because it typically develops in patients on continuous ambulatory peritoneal dialysis,[1] and is therefore usually detected by dialysis physicians. Moreover, many patients do not present with rapid symptoms,[1] and rarely visit an emergency room. However, patients with ascites for any reason can potentially develop this condition.

A physiologically negative intrathoracic pressure combined with an increased intra-abdominal pressure may open small defects in the diaphragm and promote the strictly one-way flow of ascites into the pleural space.[4,5] Similar to the way in which the vomiting reflex relates to upper endoscopy, the more rapidly the abdominal pressure increases, the more drastically the patient develops symptoms. Hence, the presence of pleural effusion resulting from a pleuroperitoneal leak should be suspected in the dyspneic non-edematous patient with history of increasing intra-abdominal pressure, and in patients in whom the effusion is only right-sided, even in the emergency room.

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CONFLICT OF INTEREST

NONE.

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